U.S. Department of Agriculture

APHIS-PPQ

APHIS 81-49
September 1986

PESTS NOT KNOWN TO OCCUR IN THE UNITED STATES OR OF LIMITED DISTRIBUTION NO. 76: A FRUIT BROWN ROT

Prepared by L. W.H. Chang, Biological Assessment Support Staff, PPQ, APHIS, USDA, Federal Building Room 634, Hyattsville, MD 20782

Disease

A FRUIT BROWN ROT

Pathogen

Teleomorph (ascigerous or perfect stage)
Monilinia fructigena Honey in Whetzel
Sclerotinia fructigena Aderhold & Ruhland
Often referred to in literature but name is invalid.

Anamorph (conidial or imperfect stage)
Monilia fructigena (Persoon) Persoon

Class: Order: Family

Ascomycetes: Helotiales: Helotiaceae

Economic Importance

M. fructigena is one of the major Monilinia pathogens of pome and stone fruits in the world; the others are M. fructicola (Winter) Honey in Whetzel, and M. laxa (Aderhold & Ruhland) Honey. Epiphytotics of M. fructigena brought on by rainy or hot weather cause severe losses. Fruit loss results from the very rapid decay of fruit on the tree and in storage, from culls of diseased fruit during harvest, and from loss of fruiting spurs that would have borne future fruit. Before development of effective fungicides, the brown rot pathogens destroyed a large part of the crop. M. fructigena has caused losses as heavy as 60 percent to pears in Denmark, and up to 38 percent to apples in the Soviet Union and the United Kingdom and to pears in Iran. In England from 1961 to 1965, sampling of one apple cultivar in refrigerated stores revealed mean losses of 0.2-1.5 percent with a range of 0.1-4.5 percent for individual orchards (Byrde and Willetts 1977, Wormald 1954).

Hosts

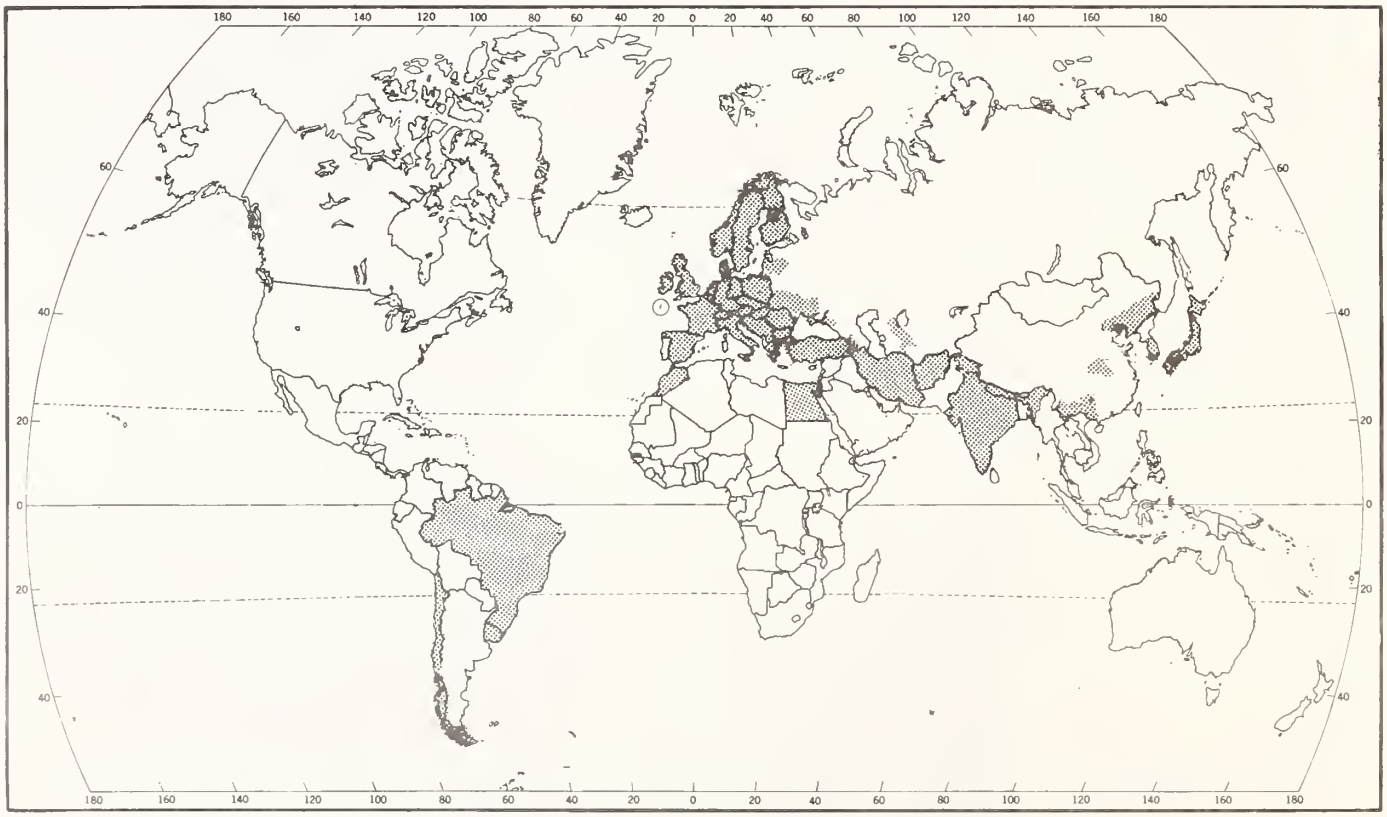
Hosts include most pome and stone fruits in the Rosaceae and a few members of other families. The following hosts were reported by Wormald (1954) unless cited otherwise: Amelanchier canadensis (Lovisolo 1956), Corylus avellana (European filbert), Cotoneaster obtusa (Negru et al. 1957), Crataegus oxyacantha (English hawthorn) (Lovisolo 1956), Cydonia oblonga (quince), Elaeagnus macrophylla, Feijoa sellowiana (feijoa) (Kechakmadze and Kikvadze 1976) (questionable host), Malus baccata (Siberian crabapple), Malus prunifolia (Negru et al. 1957), Malus sieboldii (Lovisolo 1956), Malus sylvestris (apple), Mespilus germanica (medlar), Prunus dulcis (almond), Prunus armeniaca (apricot), Prunus avium (sweet cherry), Prunus cerasifera, Prunus cerasus (sour cherry), Prunus domestica (plum), Prunus hortulana (hortulan plum), Prunus insititia (bullace plum, damson plum), Prunus laurocerasus (cherry laurel) (Mordue 1979b), Prunus mume (Japanese apricot),

Prunus persica (peach, nectarine), Prunus pseudo-cerasus, Prunus salicina (Japanese plum) (Korean Society of Plant Protection 1972), Prunus spinosa, Pyrus communis (pear), Pyrus X purpurea, Pyrus pyrifolia (sand pear) (Korean Society of Plant Protection 1972), Rubus sp. (a blackberry) (Moore and Talboys 1953), R. occidentalis (black raspberry), Sorbus aucuparia (European mountain-ash) (Mordue 1979b), and Vitis vinifera (wine grape) (Wormald 1954).

Uncommon hosts are Cornus mas (Cornelian cherry) and Ficus sp. (fig) reported by Lovisolo (1956), Berberis vulgaris (European barberry) by Negru et al. (1957), and Diospyros sp. (persimmon), Eriobotrya japonica (loquat), Fragaria sp. (strawberry), and Vaccinium sp. by Mordue (1979b).

General Distribution

Distributed throughout Europe and Asia and parts of Africa and South America. Unless cited otherwise, the Commonwealth Mycological Institute (1976) listed the following countries:



Monilinia fructigena distribution map (Prepared by Non-Regional Administrative Operations Office and Biological Assessment Support Staff, PPQ, APHIS, USDA).

Afghanistan, Austria, Belgium, Brazil, Bulgaria, Chile, China (Honan, Yunnan, Wormald (1954) reported southern Manchuria), Cyprus, Czechoslovakia, Denmark, East Germany, Egypt, Finland, France, Greece, Hungary, India, Iran, Ireland, Israel, Italy (includes Sicily), Japan, Morocco, Nepal, Netherlands, Norway, Poland, Rumania, South Korea, western Soviet Union (Latvian, Lithuanian, and Ukrainian S.S.R., Wormald (1954) reported Uzbek S.S.R. and Russian S.F.S.R. (Krasnodar region) and Teterevnikova-Babayan (1981) reported Armenian S.S.R.), Spain, Sweden, Switzerland, Turkey, United Kingdom (includes Channel Islands and Jersey), Uruguay, West Germany, and Yugoslavia.

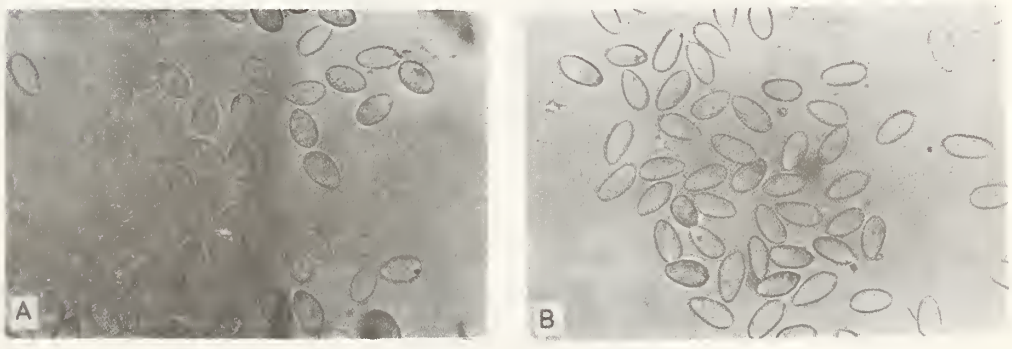
Characters

ASCIGEROUS STAGE - Rare in field. Apothecia arise from mummified, overwintered fruit. Disk diameter 3-5 mm, yellow brown then gray, concave then flattened but umbilicate. Receptacle cup-shaped; stalk cylindrical, 5-15 X 1 mm. Asci cylindrical, indistinctly stalked, broadly rounded above, pore not blue in iodine, 120-180 X 9-12 µm, 8-spored. Ascospores obliquely uniseriate, elliptical, ovoid, or slightly inequilateral, 9-12.5 X 5-6.8 µm. Paraphyses cylindrical, obtuse apex slightly enlarged to 2.5 µm (Dennis 1956).

Apothecia, asci, and ascospores are similar for the three species. Ascospores of M. fructigena (Fig. 1A), however, generally have pointed ends and lack oil drops, while ascospore ends are rounded and oil drops present in M. fructicola (Fig. 1B) and M. laxa (Harrison 1935, Matheny 1913).

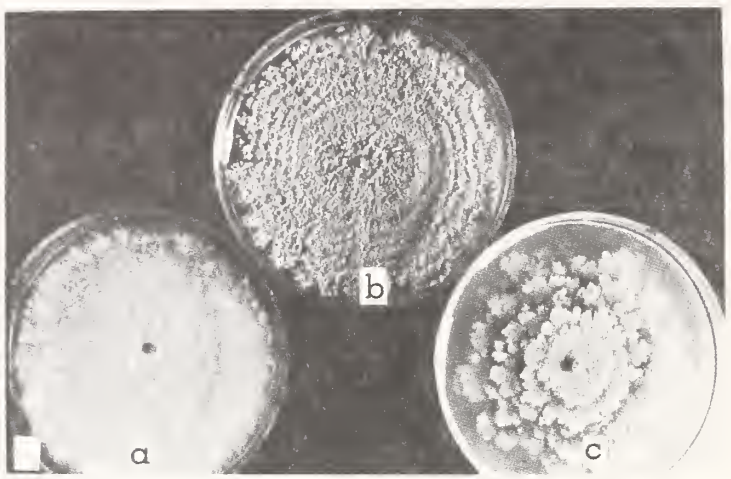
CONIDIAL STAGE - Colony margin even (Fig. 2A), mycelium in concentric rings of flat, dense, sterile tissue alternating with light buff, sporogenous tissue on potato dextrose agar under alternating light and darkness (Byrde and Willetts 1977); zonation best on medium buffered between pH 4 and 5 (Hall 1933). Mycelium usually darkens with age; or small discoid

(Fig. 1)



Monilinia ascospores. A. M. fructigena. B. M. fructicola (From Harada 1977).

(Fig. 2)



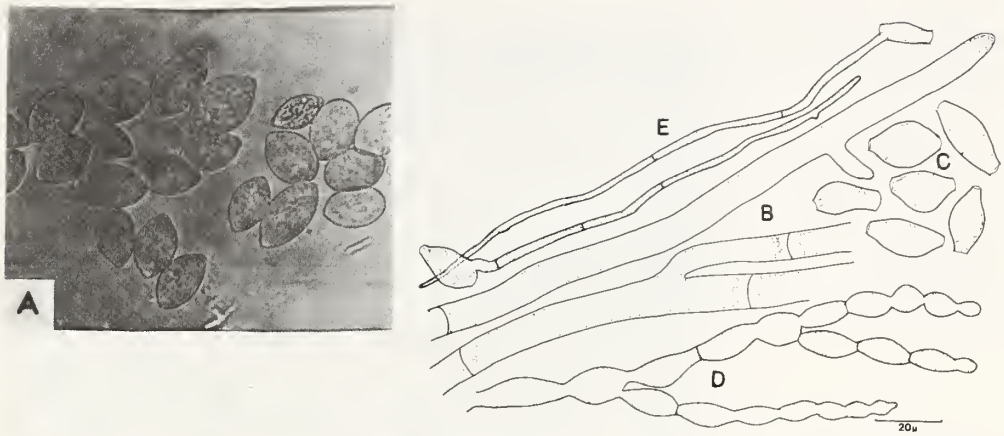
Monilinia colonies on potato sucrose agar A. M. fructigena. B. M. fructicola. C. M. laxa (From Harada 1977).

sclerotia, irregular stromatal flakes, or crusts sometimes develop on or within the medium; stromata and spermatia infrequent; large, black microconidial clusters rarely visible (Byrde and Willetts 1977). Conidia (Fig. 3A) in mass buff, elliptical, hyaline, size 12-34 X 6-15 μm (averaging about 21 X 13 μm). Germ tube (Fig. 3E) single, long before branching (Mordue 1979b).

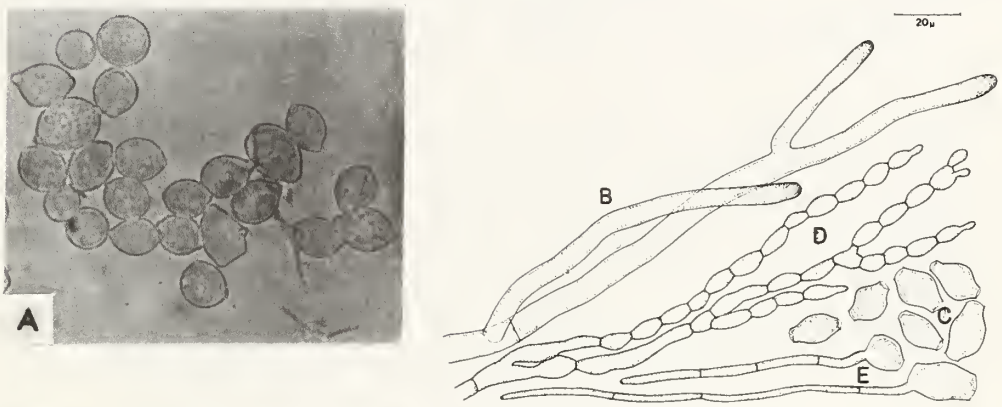
Harada (1977) observed mycelial growth on potato sucrose agar between 1 to 30° C (optimum 25° C), and maximum sporulation (albeit poor) between 5 to 30° C (optimum 15° C), at 52 percent or less relative humidity, and under alternating light and darkness (12-hour photoperiod).

The three species are compared in Table 1. These characters are not always reliable for identification because features for the three species can vary under different culture conditions and among different isolates for a species. The following two features separated the species for the isolates studied when traditional means failed. The first used the occurrence of hyphal anastomosis between germ tubes. Anastomosis commonly occurs in M. fructicola but rarely does in M. laxa (Ogawa and English 1954, Penrose et al. 1976); they did not study M. fructigena. The second used pectolytic enzyme patterns obtained by column isoelectric focusing to help separate these species (Willetts et al. 1977).

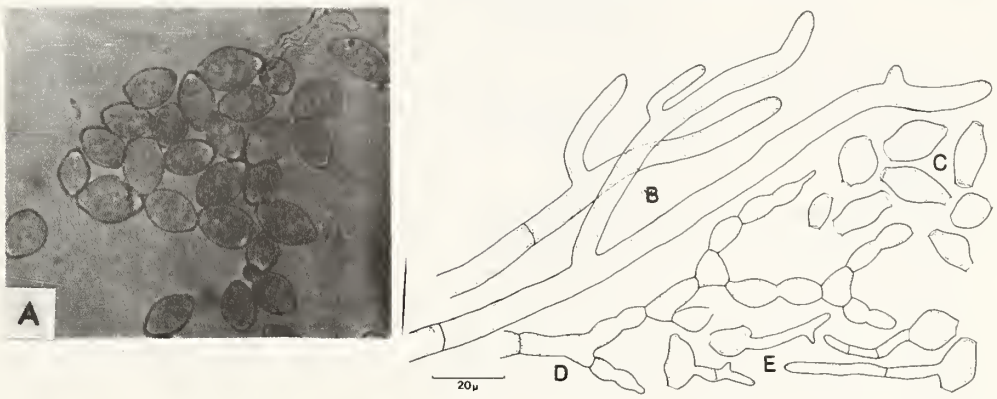
(Fig. 3)



(Fig. 4)



(Fig. 5)



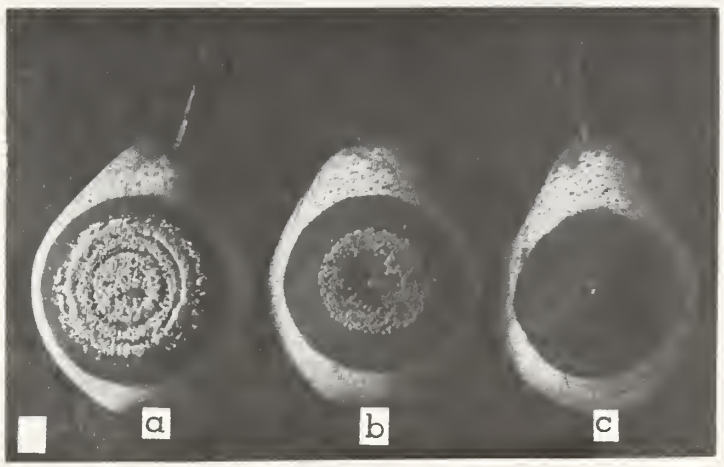
Monilinia. 3. M. fructigena. 4. M. fructicola. 5. M. laxa. A. Conidia. B. Hyphae from advancing edge of colony. C, D, E. Mature, developing, and germinating conidia (A from Harada 1977; B through E from Mordue 1979b, a, and c respectively).

Table 1. Distinguishing symptoms and conidial stages of Monilinia (Monilia) spp. for fruit brown rots. Measurements (except for pustule diameter) in micrometers (Byrde and Willetts 1977; Mordue 1979a, b, c; Willetts 1968; Wormald 1954)

	fructigena	fructicola	laxa
On Host			
Symptoms generally on	Pome and stone fruit: fruit rot, spur blight, twig canker	Stone fruit: fruit rot, blossom blight, branch canker	Stone fruit, apple, quince: blossom and twig blight, fruit rot but not of apple
Pustule development	All infected parts 15-25° C	All infected parts	Infected twigs from 5° C
Pustule diameter (mm)	1-1.5	0.4-0.8	0.4-0.8
In Culture			
Colony margin (Fig. 2)	Even	Even	Lobed
Mycelial growth	Basket-weave pattern, branching and anastomosis infrequent	Basket-weave pattern, much dichotomous branching, anastomosis frequent	Often scorpioid, geniculate, anastomosis infrequent
Conidia, stromata, spermatia	Less abundant	More abundant	Less abundant
Conidial size (average)	12-34 X 6-15 (21 X 13)	8-28 X 5-19 (15 X 9)	5-23 X 4-16 (15 X 9)
Germ tube length before branching (Figs. 3E-5E)	Long, 400-1200	Long, 200-750	Short, close to conidium
Primary hyphal diameter	7-11	7-10	6-11

Characteristic Damage: The pathogen causes a fruit rot of pome and stone fruits, black apple in storage, spur blight and twig canker of apple and peach trees, and nut rot and drop of filbert.

(Fig. 6)



Monilinia fruit rot of inoculated pear. A. M. fructigena. B. M. fructicola. C. M. laxa (From Harada 1977).

M. fructigena affects fruit at any stage from fruit set into storage. A brown spot develops at the infection site, rapidly enveloping the fruit. Many blisterlike swellings appear. The blisters assume a pattern of concentric rings (Fig. 6) on pome fruits. The blisters later burst, showing buff cushionlike tufts. Diseased fruit may stick in clusters, and either cling to the tree or drop to the ground. The relatively dry rot occurs in 1-2 days. Then the rotten fruit mummifies (Wormald 1954) to a dark, hollow, sclerotial, leathery sphere enclosing the unrotted core (Byrde and Willetts 1977).

Black apples develop in storage, sometimes in the field. Rot begins at the point of injury or a stalkless end. The entire fruit remains firm and turns a smooth shiny black, later wrinkling. Spore tufts are rare (Wormald 1954).

Symptoms in other plant parts originate from diseased fruit, never from diseased flowers or direct infection of woody parts. The resultant dead fruiting spurs and cankers (localized lesions) on the branches may carry buff spore tufts. The brown, collapsed tissues extend up and down from the penetration site and may exude gum (Byrde and Willetts 1977, Wormald 1954). Dead twigs or branches can be traced to a girdled site (Wormald 1954).

Damage to filbert begins in the soft shell stage (Moore 1947). Bracts discolor and wither. Shells turn brown, especially at the base. Infection of one or more nuts in a cluster may cause the entire cluster to drop (Wormald 1944) within a week. The nuts turn brown and completely rot. Buff conidial tufts develop on the outside of the shell (Moore 1947, 1950).

Isolated damage has been reported of lesions on peach shoots, infected leaves, experimental infection of bud unions, crown dieback of apricot (Wormald 1954) and core rot in apple (Dowson 1926).

Generally of the three Monilinia species, M. fructigena causes more serious injury to pome than to stone fruit, M. fructicola causes more fruit rot of stone than of pome fruit, and M. laxa induces a more severe blossom and twig blight. Dead woody tissue can be traced to a dead flower in the last two diseases, but not in the first. See Table 1.

Detection Notes

Movement of fruits or nursery stock infected by M. fructigena could introduce this pest into new areas. Fresh fruits may enter the United States only under USDA permit and are subject to inspection as specified in Title 7, Part 319.56 of the Code of Federal Regulations. Propagative material (including cut flowers) of its primary hosts (Malus, Pyrus, Prunus, Chaenomeles, Cydonia spp.) are prohibited by Part 319.37 from most countries except under departmental permit for scientific purposes, and are subject to stringent entry requirements from certain other countries. Other hosts are subject to entry requirements, such as postentry quarantine, to exclude the pathogen.

Combined with these efforts to prevent entry, PPQ has intercepted M. fructigena 157 times from 1974 to 1984 at U.S. ports of entry. Interceptions were made on fruit (except for one interception on a leaf) of Malus spp., M. sylvestris (apple), Prunus spp., P. avium (sweet cherry), P. domestica (plum), and P. persica (peach) in baggage (82 times), holds (1), quarters (4), and stores (64). Countries of origin included Belgium, France, Greece, Hungary, Italy, Japan, Netherlands, Spain, Switzerland, United Kingdom, West Germany, and Yugoslavia. Interceptions from Portugal and Trinidad and Tobago (countries not specifically cited in the literature) may represent transshipments from other areas.

Survey for the following features.

1. Examine pome and stone fruits for diseased fruits or mummies on the tree or the ground. Blisters or buff spore cushions may be in concentric rings. Diseased fruit will be firm and relatively dry, not soft and wet. Several fruits may stick to each other on the tree, ground, or in packing. Look for firm, glossy black apples in storage.

2. Examine dead woody tissue such as fruit spurs, twigs, and branches. Look for cankers, some with gum exudate. Trace the sunken brown tissue from the fruit or the pedicel (never the flower) through the spur and onward to the twig and branch. Dead tissue extends outward on both sides of the penetration site. Dead twigs or branches can be traced to a girdled site. The diseased parts may carry buff spore tufts.

3. On filberts, look for shrunken, discolored bracts; brown shells, especially at the base; fallen nut clusters; brown, rotten nuts, and buff conidial tufts on the husk.

Submit suspect material for identification. To prevent disease dissemination, ship specimens in sealed double containers (one inside the other) with screw tops.

Biology and Etiology

M. fructigena overwinters as mycelium in dead diseased host tissues, such as fruit mummies, twigs, peduncles, and cankers on branches. Such mycelium survives long periods of unfavorable environmental conditions better than the spores do, making the mycelium more important for survival (Byrde and Willetts 1977) although conidia of M. fructigena resist uniform extreme dessication in vitro (Naqvi and Good 1957). Mycelium in a mummy was still viable after 10 years (Gram and Weber 1953).

With suitable spring temperatures, day length, and relative humidity, this overwintered mycelium produces primary inoculum on the surface of mummies (Byrde and Willetts 1977), cankers, and other blighted twigs (Mordue 1979b). Abundant tufts of conidiophores emerge and bear chains of conidia as the imperfect (anamorph) or Monilia stage. A new crop of conidia is produced during the season whenever conditions become favorable. One mummy could release up to 4 million spores (Byrde 1952).

The mummy must absorb a minimum of 21 percent moisture to sporulate at 26° C. At 20° C, sporulation occurs about 12 hours after host tissue is water soaked. Spore production occurs on dry tissues (such as peduncles, laterals, and mummies) only during wet weather, but it will occur on moist tissues (such as young and ripe fruits) in a dry atmosphere (Byrde and Willetts 1977).

Another source of primary inoculum is the apothecium. Only mummies that have overwintered on the ground sometimes produce apothecia, which in turn release ascospores. This spore type is the perfect (teleomorph) or Monilinia stage. Conditions favoring apothecial production usually lead to the breakdown of the mummy, making apothecial production possible only for a

year. The function of a third type of spore, the microconidium, is unknown; many are produced in small cavities and on the surface of fruit mummies (Byrde and Willetts 1977).

Wind and air currents, water splash, or bird and insect vectors disperse the spores (Byrde and Willetts 1977) to the host. Air currents can carry conidia long distances (Wormald 1954).

With a free film of moisture (such as water or juice exuded by injured fruit) on the host, conidia germinate in 1 hour under optimal conditions; ascospores require 4-6 hours. The germ tube can penetrate fruit at any stage of fruit development although ripening fruit is more susceptible than green fruit (Byrde and Willetts 1977). Once the fungus penetrates, it rapidly spreads through the fruit, so that in 4-5 days conidiophores erupt through the surface. These produce conidia, providing inoculum for secondary infection of fresh host fruit (Byrde and Willetts 1977, Wormald 1954). The infection cycle is repeated several times during the season on fresh host fruit.

Apple blossoms can be infected, but flowers and conidia rarely occur together. When rains extended blossoming to coincide with spore ripening, infection of blossoms occurred (Wormald 1954).

Secondary infection also occurs from infected tissues. Hyphae from the diseased fruit may invade the unbroken skin of adjacent fruit where they touch (Byrde and Willetts 1977, Wormald 1954), thus infecting all fruit in the cluster. Hyphae may also invade woody tissues by spreading from the fruit through the fruit stalk (pedicel), into the fruit spur (peduncle), and from there into the branch, resulting in cankers (Byrde and Willetts 1977; Wormald 1954). Stem cankers seldom develop from spores penetrating injured woody tissue (Byrde and Willetts 1977). Infection in branches of apple, plum, and peach expands rapidly and then stops in a few weeks, sealed off by callus. If the canker girdles the branch, the terminal dies (Wormald 1954).

With sporulation, the diseased fruit dries, shrinks, mummifies, and either clings to the tree or drops to the ground. The diseased twig dies. Mycelium survives in this host tissue to initiate the disease cycle the following spring.

Threshholds for some factors affecting viability are known. Conidia retained some viability after 6 months of storage at -14 to -18° C, yet lost it after less than 3 months of overwintering outdoors (Wormald 1954). Spores survived 10 months at 5° C and 75 percent relative humidity, and 5 months at 35° C. Viability was unaffected by months of storage at humidities ranging from 15 to 75 percent (Naqvi and Good 1957).

For mycelium, growth continued slowly at 0° C, optimally at 25° C, stopped at 35° C, and died at about 50° C (Byrde and Willetts 1977, Wormald 1954).

Rots, therefore, continue in cold storage and proceed rapidly in the field with warm temperatures. Temperatures near freezing merely retard growth (Wormald 1954).

Cloudy weather that is wet or misty favors production, dispersal, and germination of conidia. Many generations rapidly develop and build up during spring and summer with wet, cloudy weather resulting in epiphytotics (Wormald 1954). A dry climate, however, may not prevent outbreaks. A semiarid area of New South Wales undergoes epiphytotics incited by M. fructicola because of the activities of oriental fruit moth (Grapholita molesta (Busck)) as a wounding agent and dried fruit beetles (Carpophilus spp.) as vectors (Kable 1969).

Although the pathogen can penetrate apples through the lenticels (Horne 1933), injuries to fruit increase infection incidence. Fruit injury occurs in various ways. Openings can be caused by alternating wet and dry periods (especially a drought), hail, rubbing by other plant parts, infection by other pathogens, insect egg laying or feeding (such as Curculio nucum L. (nut weevil) in filbert (Moore 1947)), bird feeding, severe russeting from sprays, or careless harvesting resulting in bruised or stalkless fruits (Wormald 1954).

Control

Removal and destruction of diseased plant parts (including those on nearby ornamental or wild hosts) reduce the source of primary inoculum and thus reduce disease buildup during the season. Although airborne spores will still incite the disease in a clean orchard, infections are more prevalent where the inoculum source is already in the orchard. Even in a mild disease year, removal of the small amount of diseased material is essential. Otherwise, inoculum rapidly increases with favorable weather to epiphytotic proportions (Wormald 1954).

Besides sanitation, other preventive practices reduce disease incidence. Avoid interplanting of susceptible cultivars to prevent a domino effect of inoculum building up on successively ripening cultivars (Kable 1971). Minimizing fruit injury by insects, other pathogens, rubbing twigs, and careless picking help limit the number of infection sites (Wormald 1954).

Byrde and Willetts (1977) discuss possible chemical controls to eliminate winter inoculum, inhibit spore germination, and inhibit sporulation during the growing season, as well as protective preharvest sprays.

Byrde and Willetts (1977) and Gram and Weber (1953) list several cultivars of apple, pear, and plum that are less susceptible to M. fructigena.

Literature Cited

Byrde, R. J. W. Experiments on the control of brown rot of apples and plums. II. Winter spraying trials. J. Hortic. Sci. 27(3):192-200; 1952.

Byrde, R. J. W.; Willetts, H. J. The brown rot fungi of fruit: their biology and control. New York: Pergamon Press; 1977.

Commonwealth Mycological Institute. Distribution maps of plant diseases. No. 22. London: Commonwealth Agricultural Bureaux; 1976.

Dennis, R. W. G. A revision of the British Helotiaceae in the Herbarium of the Royal Botanic Gardens, Kew, with notes on related European species. Mycological Papers 62. Kew: Commonwealth Mycological Institute; 1956: 140-144.

Dowson, W. J. On a core rot and premature fall of apples associated with Sclerotinia fructigena. Trans. Br. Mycol. Soc. 11(1-2):155-161; 1926.

Gram, E.; Weber, A. Plant diseases in orchard, nursery and garden crops. New York: Philosophical Library; 1953: 121-123, 161, 193. Translated from the Danish by E. Ramsden.

Hall, M. P. An analysis of the factors controlling the growth form of certain fungi, with especial reference to Sclerotinia (Monilia) fructigena. Ann. Bot. 47(187):543-578; 1933.

Harada, Y. Studies on the Japanese species of Monilinia (Sclerotiniaceae). Bull. Fac. Agric. Hirosaki Univ. 27:30-109; 1977 (In Japanese, English summary).

Harrison, T. H. Brown rot of fruits and associated diseases of deciduous fruit trees. II. The apothecia of the causal organisms. J. Proc. R. Soc. New South Wales 68(2):154-176; 1935.

Horne, A. S. Biological work on fruit. In: Report of the food investigation board for the year 1932, Department of Scientific and Industrial Research. London: His Majesty's Stationery Office; 1933: 279, 285-286, 288.

Kable, P. F. Brown rot of stone fruits on the Murrumbidgee irrigation areas. I. Aetiology of the disease in canning peaches. Aust. J. Agric. Res. 20(2):301-316; 1969.

Kable, P. F. Better control measures for brown rot of stone fruits. Agric. Gaz. New South Wales 82(3-4):194-201; 1971.

Kechakmadze, L. A.; Kikvadze, I. V. Diseases of feijoa. Zashch. Rast. 8:43; 1976 (In Russian).

Korean Society of Plant Protection. A list of plant diseases, insect pests, and weeds in Korea. Seoul, South Korea: Korean Society of Plant Protection; 1972.

Lovisolo, O. Le Monilie dei fruttiferi. Boll. Stn. Patol. Veg. Ser. 3, 13:7-40; 1956 (English summary).

Matheny, W. A. A comparison of the American brown-rot fungus with Sclerotinia fructigena and S. cinerea of Europe. Bot. Gaz. 56(5):418-432; 1913.

Moore, M. H. Preliminary report on Monilia fructigena and Botrytis cinerea as wound parasites of cobnuts and filberts. Annu. Rep. E. Malling Res. Stn. for 1946, p. 120-121; 1947.

_____. Nut rotting in Corylus avellana L. in relation to the activities of the nut weevil, Balaninus nucum L. J. Hortic. Sci. 25(4):213-224; 1950.

Moore, M. H.; Talboys, P. W. Note on infection of blackberry fruits by the brown rot fungus, Monilia fructigena. Annu. Rep. E. Malling Res. Stn. for 1952, p. 136-137; 1953.

Mordue, J. E. M. CMI descriptions of pathogenic fungi and bacteria. No. 616. Sclerotinia fructicola. London: Commonwealth Agricultural Bureaux; 1979a.

_____. CMI descriptions of pathogenic fungi and bacteria. No. 617. Sclerotinia fructigena. London: Commonwealth Agricultural Bureaux; 1979b.

_____. CMI descriptions of pathogenic fungi and bacteria. No. 619. Sclerotinia laxa. London: Commonwealth Agricultural Bureaux; 1979c.

Naqvi, S. H. Z.; Good, H. M. Studies of the aging of conidia of Monilinia fructicola (Wint.) Honey. I. Germination rates and longevity. Can. J. Bot. 35(5):635-645; 1957.

Negru, A.; Mircea, E.; Crisan, A. Plante gazde noi pentru Monilia fructigena Pers. Studii Cercet. Agron. Acad. Repub. Pop. Rom., Cluj 8(1-2):93-98; 1957 (Russian and French summaries).

Ogawa, J. M.; English, H. Means of differentiating atypical isolates of Sclerotinia laxa and S. fructicola. Phytopathology 44(9):500; 1954.

Penrose, L. J.; Tarran, J.; Wong, A.-L. First record of Sclerotinia laxa Aderh. & Ruhl. in New South Wales: differentiation from S. fructicola (Wint.) Rehm. by cultural characteristics and electrophoresis. Aust. J. Agr. Res. 27(4):547-556; 1976.

Teterevnikova-Babayan, D. N. Some data on fungus diseases of rare and disappearing plants of the Armenian SSR. Biol. Zh. Armenii 34(11):1099-1106; 1981 (In Russian; Armenian, English summaries). Taken from: Rev. Plant Pathol. 61(11):537; 1982.

Willetts, H. J. The development of stromata of Sclerotinia fructicola and related species. I. In culture. Trans. Br. Mycol. Soc. 51(5):625-632, Plates 47-48; 1968.

Willetts, H. J.; Byrde, R. J. W.; Fielding, A. H.; Wong, A.-L. The taxonomy of the brown rot fungi (Monilinia spp.) related to their extracellular cell wall-degrading enzymes. J. Gen. Microbiol. 103(1):77-83; 1977.

Wormald, H. Nut drop: a disease of cultivated hazel nuts. Gard. Chronicle Ser. 3, 115(2980):60-61; 1944.

_____. The brown rot diseases of fruit trees. Ministry of Agriculture and Fisheries (United Kingdom). Technical Bulletin No. 3. London: Her Majesty's Stationery Office; 1954.